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=> d his

     (FILE 'HOME' ENTERED AT 15:34:55 ON 21 JUL 2003)

     INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI,
     BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA,
     CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB,
     DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...' ENTERED AT 15:35:36 ON
     21 JUL 2003
               SEA (ADIPONECTIN) OR (ADIPONECTIN-LIKE)
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                 4   FILE ADISCTI
                 1   FILE ADISINSIGHT
                 3   FILE ADISNEWS
                 7   FILE AGRICOLA
               218   FILE BIOSIS
                 4   FILE BIOTECHABS
                 4   FILE BIOTECHDS
                53   FILE BIOTECHNO
                35   FILE CABA
                24   FILE CANCERLIT
               280   FILE CAPLUS
                 1   FILE CIN
                 5   FILE CONFSCI
                25   FILE DDFU
                22   FILE DGENE
                 1   FILE DRUGNL
                28   FILE DRUGU
                 2   FILE DRUGUPDATES
                32   FILE EMBAL
               198   FILE EMBASE
               135   FILE ESBIOBASE
                 2   FILE FEDRIP
                 1   FILE FROSTI
                 8   FILE GENBANK
                 1   FILE IFIPAT
                50   FILE JICST-EPLUS
                12   FILE LIFESCI
               232   FILE MEDLINE
                68   FILE PASCAL
                 1   FILE PHAR
                 3   FILE PHIN
                 2   FILE PROMT
               258   FILE SCISEARCH
               100   FILE TOXCENTER
                 4   FILE USPATFULL
                 6   FILE WPIDS
                 6   FILE WPINDEX
L1               QUE (ADIPONECTIN) OR (ADIPONECTIN-LIKE)
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     FILE 'CAPLUS, SCISEARCH, MEDLINE, BIOSIS, EMBASE, ESBIOBASE, TOXCENTER,
     PASCAL, BIOTECHNO, JICST-EPLUS' ENTERED AT 15:37:00 ON 21 JUL 2003
L2           226 S L1 AND (ISOLAT? OR CHARACT? OR PURIF?)
L3            19 S L2 AND PURIF?
L4            11 DUP REM L3 (8 DUPLICATES REMOVED)
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=> d l4 ibib ab 1-11

L4 ANSWER 1 OF 11 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STNDUPLICATE 1
ACCESSION NUMBER: 2003:472814 SCISEARCH
THE GENUINE ARTICLE: 683UD
TITLE: Involvement of AMP-activated protein kinase in glucose uptake stimulated by the globular domain of **adiponectin** in primary rat adipocytes
AUTHOR: Wu X D; Motoshima H; Mahadev K; Stalker T J; Scalia R; Goldstein B J (Reprint)
CORPORATE SOURCE: Thomas Jefferson Univ, Jefferson Med Coll, Div Endocrinol Diabet & Metab Dis, Dorrance H Hamilton Res Labs, Dept Med, Rm 349 Alumni Hall, 1020 Locust St, Philadelphia, PA 19107 USA (Reprint); Thomas Jefferson Univ, Jefferson Med Coll, Div Endocrinol Diabet & Metab Dis, Dorrance H Hamilton Res Labs, Dept Med, Philadelphia, PA 19107 USA; Thomas Jefferson Univ, Jefferson Med Coll, Dept Physiol, Philadelphia, PA 19107 USA
COUNTRY OF AUTHOR: USA
SOURCE: DIABETES, (JUN 2003) Vol. 52, No. 6, pp. 1355-1363. Publisher: AMER DIABETES ASSOC, 1701 N BEAUREGARD ST, ALEXANDRIA, VA 22311-1717 USA. ISSN: 0012-1797.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 59

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Adiponeetin is an abundant adipocyte-derived plasma protein with anti-atherosclerotic and insulin-sensitizing properties that, suppresses hepatic glucose production and enhances glucose uptake into skeletal muscle. To **characterize** the potential effects of **adiponectin** on glucose uptake into adipose cells, we incubated **isolated** epididyinal rat adipocytes with the globular domain of recombinant **adiponectin purified** from an E. coli expression,system. Globular **adiponectin** increased glucose uptake in adipocytes without stimulating tyrosine phosphorylation of the insulin receptor or insulin receptor substrate-1, and without enhancing phosphorylation of Akt on Ser-473. Globular **adiponectin** further enhanced, insulin-stimulated glucose uptake at submaximal insulin concentrations and reversed the inhibitory effect of tumor necrosis,factor-a on insulin-stimulated glucose uptake. Cellular treatment with globular **adiponectin** increased the Thr-172 phosphorylation and catalytic activity of AMP-activated protein kinase and enhanced the Ser-79 phosphorylation of acetyl CoA carboxylase; an enzyme downstream of AMP kinase in Adipose cells. Inhibition of AMP kinase activation using two pharmacological inhibitors (adenine 9-beta-D-axabinofuranoside and compound C) completely abrogated the increase in glucose uptake. stimulated by globular **adiponectin**, indicating that AMP kinase is integrally involved in the **adiponectin** signal transduction pathway. Coupled with recent evidence that the effects of **adiponectin** are mediated via AMP kinase activation in liver and skeletal muscle; the findings reported here provide an important mechanistic link in the signaling effects of **adiponectin** in diverse metabolically responsive tissues.

L4 ANSWER 2 OF 11 MEDLINE on STN
ACCESSION NUMBER: 2003041047 MEDLINE
DOCUMENT NUMBER: 22436055 PubMed ID: 12547549
TITLE: **Adiponectin** and protection against type 2 diabetes mellitus.
COMMENT: Erratum in: Lancet. 2002 Mar 22;361(9362):1060
AUTHOR: Spranger Joachim; Kroke Anja; Mohlig Matthias; Bergmann Manuela M; Ristow Michael; Boeing Heiner; Pfeiffer Andreas

F H
CORPORATE SOURCE: Department of Nutrition, Endocrinology, and Metabolism, Benjamin Franklin Medical Centre, Free University Berlin, Germany.. spranger@mail.dife.de
SOURCE: LANCET, (2003 Jan 18) 361 (9353) 226-8.
Journal code: 2985213R. ISSN: 0140-6736.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 20030129
Last Updated on STN: 20030410
Entered Medline: 20030205

AB **Adiponectin** is an adipocyte-derived peptide, which has anti-inflammatory and insulin-sensitising properties. We designed a nested case-control study to assess whether baseline **adiponectin** concentrations in plasma are independently associated with risk of type 2 diabetes. We found that **adiponectin** concentrations in plasma were lower among individuals who later developed type 2 diabetes than among controls (mean 5.34 microg/mL [SD 3.49] vs 6.87 microg/mL [4.58], p<0.0001). High concentrations of **adiponectin** were associated with a substantially reduced relative risk of type 2 diabetes after adjustment for age, sex, waist-to-hip ratio, body-mass index, smoking, exercise, alcohol consumption, education, and glycosylated haemoglobin A(1c) (odds ratio 4th vs 1st quartile 0.3 [95% CI 0.2-0.7], p=0.0051). We conclude that **adiponectin** is independently associated with a reduced risk of type 2 diabetes in apparently healthy individuals.

L4 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2002:646135 CAPLUS
DOCUMENT NUMBER: 137:346864
TITLE: Oligomerization state-dependent activation of NF-.kappa.B signaling pathway by adipocyte complement-related protein of 30 kDa (Acrp30)
AUTHOR(S): Tsao, Tsu-Shuen; Murrey, Heather E.; Hug, Christopher; Lee, David H.; Lodish, Harvey F.
CORPORATE SOURCE: Whitehead Institute for Biomedical Research, Cambridge, MA, 02142, USA
SOURCE: Journal of Biological Chemistry (2002), 277(33), 29359-29362
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Adipocyte complement-related protein of 30 kDa (Acrp30)/ **adiponectin** is an adipocyte-derived hormone that affects lipid and glucose metab. in muscle and liver, but its phys. and biochem. properties are poorly **characterized**. Here we have used several approaches to show that Acrp30 expressed in and **purified** from Escherichia coli and human embryonic kidney 293T cells forms trimers and hexamers; 293T cells also produce a higher mol. wt. species. Similar Acrp30 oligomers were found in mouse serum as well as in 3T3-L1 adipocyte-conditioned medium, although in different proportions. In parallel, we assessed whether Acrp30 is a signaling mol. by searching for promoter or enhancer elements that respond to Acrp30 or its **isolated** trimeric globular C-terminal domain, gAcrp30. Acrp30 addn. to C2C12 myocytes or myotubes led to activation of NF-.kappa.B transcription factor in a manner dependent upon phosphorylation and degrdn. of I.kappa.B-.alpha.. Importantly, only hexameric and larger isoforms of Acrp30 activated NF-.kappa.B; trimeric Acrp30 or gAcrp30 could not activate NF-.kappa.B. Our data indicate that oligomerization of Acrp30 is important for at least some of its biol. activities, and changes

in the relative abundance of each oligomeric isoform in plasma may regulate Acrp30 activity.
REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 4 OF 11 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 2002:526350 SCISEARCH
THE GENUINE ARTICLE: 557XP
TITLE: **Purification** of globular form of **adiponectin** in human serum and its potential role in insulin sensitivity
AUTHOR: Waki H (Reprint); Yamauchi T; Kamon J; Ito Y; Uchida S; Oike Y; Yamamura K; Kimura S; Kadowaki T
SOURCE: DIABETES, (JUN 2002) Vol. 51, Supp. [2], pp. A455-A455. MA 1871.
Publisher: AMER DIABETES ASSOC, 1660 DUKE ST, ALEXANDRIA, VA 22314 USA.
ISSN: 0012-1797.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0

L4 ANSWER 5 OF 11 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 2001:647994 SCISEARCH
THE GENUINE ARTICLE: 461BX
TITLE: Identification and adipocyte differentiation-dependent expression of the unique disialic acid residue in an adipose tissue-specific glycoprotein, adipo Q
AUTHOR: Sato C; Yasukawa Z; Honda N; Matsuda T; Kitajima K (Reprint)
CORPORATE SOURCE: Nagoya Univ, Grad Sch Bioagr Sci, Dept Appl Mol Biosci, Nagoya, Aichi 4648601, Japan (Reprint); Nagoya Univ, Biosci Ctr, Div Oncogenesis, Dept Anim Sci, Nagoya, Aichi 4648601, Japan
COUNTRY OF AUTHOR: Japan
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (3 AUG 2001) Vol. 276, No. 31, pp. 28849-28856.
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
ISSN: 0021-9258.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 34
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Recently, we have shown that alpha2,8-linked disialic acid (diSia) residue occurs in glycoproteins more frequently than ever recognized (Sato, C., Fukuoka, H., Ohta, K., Matsuda, T., Koshino, R., Kobayashi K., Troy, F. A., II, and Kitajima, K. (2000) J. BioL Chem. 275,15422-15431). In the course of identification of the diSia-containing glycoproteins in mammals, the 30-kDa glycoprotein was found in bovine serum. The 30-kDa glycoprotein was shown to be the bovine adipo Q, an adipocyte-specific protein, based on the partial amino acid sequences and the immuno-cross-reactivity with the recombinant mouse adipo Q. The bovine adipo, Q was shown to have no N-linked but O-linked glycan(s) containing the diSia epitope, Neu5Ac alpha2-8Neu5Ac alpha2 --> 3Gal. Furthermore, the diSia epitope was also found in the mouse adipo Q in serum as well as in the 3T3-L1 cells that are fully differentiated into adipocytes. Notably, among the known alpha2,8-sialyltransferases, only the alpha2,8-sialyltransferase III mRNA was detected in the 3T3-L1 cells at any stages of differentiation, and the recombinant alpha2,8-sialyltransferase III could sialylate the **purified** bovine adipo Q. Thus, this study clearly provides the new findings that adipo Q is the diSia-containing glycoprotein and a physiological substrate of alpha2,8-sialyltransferase III, whose substrates have not been identified so far.

L4 ANSWER 6 OF 11 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STNDUPLICATE 3
ACCESSION NUMBER: 2002:21442 SCISEARCH
THE GENUINE ARTICLE: 503WC
TITLE: Endogenous glucose production is inhibited by the adipose-derived protein Acrp30
AUTHOR: Combs T P; Berg A H; Obici S; Scherer P E; Rossetti L (Reprint)
CORPORATE SOURCE: Albert Einstein Coll Med, Dept Pharmacol, 1300 Morris Pk Ave, Bronx, NY 10461 USA (Reprint); Albert Einstein Coll Med, Dept Cell Biol, Bronx, NY 10461 USA; Albert Einstein Coll Med, Dept Med, Bronx, NY 10461 USA; Albert Einstein Coll Med, Ctr Diabet Res & Training, Bronx, NY 10461 USA; Albert Einstein Coll Med, Dept Mol Pharmacol, Bronx, NY 10461 USA
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF CLINICAL INVESTIGATION, (DEC 2001) Vol. 108, No. 12, pp. 1875-1881.
Publisher: AMER SOC CLINICAL INVESTIGATION INC, 35 RESEARCH DR, STE 300, ANN ARBOR, MI 48103 USA.
ISSN: 0021-9738.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 38

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Intraperitoneal injection of **purified** recombinant Acrp30 lowers glucose levels in mice. To gain insight into the mechanism(s) of this hypoglycemic effect, **purified** recombinant Acrp30 was infused in conscious mice during a pancreatic euglycemic clamp. In the presence of physiological hyperinsulinemia, this treatment increased circulating Acrp30 levels by approximately twofold and stimulated glucose metabolism. The effect of Acrp30 on in vivo insulin action was completely accounted for by a 65% reduction in the rate of glucose production. Similarly, glucose flux through glucose-6-phosphatase (G6Pase) decreased with Acrp30, whereas the activity of the direct pathway of glucose-6-phosphate biosynthesis, an index of hepatic glucose phosphorylation, increased significantly. Acrp30 did not affect the rates of glucose uptake, glycolysis, or glycogen synthesis. These results indicate that an acute increase in circulating Acrp30 levels lowers hepatic glucose production without affecting peripheral glucose uptake. Hepatic expression of the gluconeogenic enzymes phosphoenolpyruvate carboxykinase and G6Pase mRNAs was reduced by more than 50% following Acrp30 infusion compared with vehicle infusion. Thus, a moderate rise in circulating levels of the adipose-derived protein Acrp30 inhibits both the expression of hepatic gluconeogenic enzymes and the rate of endogenous glucose production.

L4 ANSWER 7 OF 11 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STNDUPLICATE 4
ACCESSION NUMBER: 2001:855020 SCISEARCH
THE GENUINE ARTICLE: 485EK
TITLE: The adipocyte-secreted protein Acrp30 enhances hepatic insulin action
AUTHOR: Berg A H; Combs T P; Du X L; Brownlee M; Scherer P (Reprint)
CORPORATE SOURCE: Albert Einstein Coll Med, Dept Cell Biol, Bronx, NY 10467 USA (Reprint); Albert Einstein Coll Med, Dept Med, Bronx, NY 10467 USA; Albert Einstein Coll Med, Dept Pathol, Bronx, NY 10467 USA; Albert Einstein Coll Med, Ctr Diabet Res & Training, Bronx, NY 10467 USA
COUNTRY OF AUTHOR: USA
SOURCE: NATURE MEDICINE, (AUG 2001) Vol. 7, No. 8, pp. 947-953.
Publisher: NATURE AMERICA INC, 345 PARK AVE SOUTH, NEW YORK, NY 10010-1707 USA.
ISSN: 1078-8956.

DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 33
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Acrp30 is a circulating protein synthesized in adipose tissue. A single injection in mice of **purified** recombinant Acrp30 leads to a 2-3-fold elevation in circulating Acrp30 levels, which triggers a transient decrease in basal glucose levels. Similar treatment in ob/ob, NOD (non-obese diabetic) or streptozotocin-treated mice transiently abolishes hyperglycemia. This effect on glucose is not associated with an increase in insulin levels. Moreover, in **isolated** hepatocytes, Acrp30 increases the ability of sub-physiological levels of insulin to suppress glucose production. We thus propose that Acrp30 is a potent insulin enhancer linking adipose tissue and whole-body glucose metabolism.

L4 ANSWER 8 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:278603 BIOSIS
DOCUMENT NUMBER: PREV200100278603
TITLE: **Adiponectin**, an adipocyte-specific secretory protein, inhibits B lymphopoiesis in culture.
AUTHOR(S): Yokota, Takafumi (1); Oritani, Kenji; Kouro, Taku (1); Meka, Reddy (1); Medina, Kay L. (1); Tomiyama, Yoshiaki; Matsuzawa, Yuji; Kincade, Paul W. (1)
CORPORATE SOURCE: (1) Oklahoma Medical Research Foundation, 825 Northeast 13th Street, Oklahoma City, OK, 73104 USA
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A318. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The stromal cells that support blood cell production within bone marrow are pre-adipocytes and functional interactions with marrow fat cells have long been suspected. **Adiponectin** was recently **isolated** as an adipocyte product and shown to have structural similarities to C1q as well as members of the TNF superfamily. It suppresses myeloid differentiation in short term bone marrow cultures and also inhibits macrophage functions. These observations raised the possibility that precursors of other blood cell lineages interact with fat cells in marrow via **adiponectin**. We have now determined that the factor blocks B lymphopoiesis in Whitlock-Witte type bone marrow cultures, but not the production of myeloid cells in Dexter cultures. Several observations suggest that non-lymphoid cells represent the target of this new mediator, and the B lymphoid lineage is only indirectly influenced. Highly **purified** lymphocyte precursors in stromal cell-free, serum-free cultures were unaffected by **adiponectin**. Similarly, there was no influence on IL-7 responding pro-B cells in clonal assays. The cytokine dramatically inhibited, and even reversed adipogenesis in culture, suggesting that it may normally be a feedback inhibitor of this process. PCR analyses are being conducted with cloned stromal cells that are responsive to **adiponectin**, with a view to learning if a negative regulator of B lymphopoiesis is induced. Preliminary results suggest that expression of TNFalpha, TGFbeta, interferons and a new interferon-like cytokine known as limitin are not up-regulated by **adiponectin**. Further studies should be informative about the role of fat cells within bone marrow and could reveal some involvement of **adiponectin** with lymphocyte production.

L4 ANSWER 9 OF 11 MEDLINE on STN
ACCESSION NUMBER: 2000072595 MEDLINE
DOCUMENT NUMBER: 20072595 PubMed ID: 10604883

TITLE: Novel modulator for endothelial adhesion molecules: adipocyte-derived plasma protein **adiponectin**.
AUTHOR: Ouchi N; Kihara S; Arita Y; Maeda K; Kuriyama H; Okamoto Y; Hotta K; Nishida M; Takahashi M; Nakamura T; Yamashita S; Funahashi T; Matsuzawa Y
CORPORATE SOURCE: Department of Internal Medicine and Molecular Science, Graduate School of Medicine, Osaka University, Osaka, Japan.. ouchi@imed2.med.osaka-u.ac.jp
SOURCE: CIRCULATION, (1999 Dec 21-28) 100 (25) 2473-6.
Journal code: 0147763. ISSN: 0009-7322.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000124
Last Updated on STN: 20000124
Entered Medline: 20000113

AB BACKGROUND: Among the many adipocyte-derived endocrine factors, we recently found an adipocyte-specific secretory protein, **adiponectin**, which was decreased in obesity. Although obesity is associated with increased cardiovascular mortality and morbidity, the molecular basis for the link between obesity and vascular disease has not been fully clarified. The present study investigated whether **adiponectin** could modulate endothelial function and relate to coronary disease. METHODS AND RESULTS: For the in vitro study, human aortic endothelial cells (HAECs) were preincubated for 18 hours with the indicated amount of **adiponectin**, then exposed to tumor necrosis factor-alpha (TNF-alpha) (10 U/mL) or vehicle for the times indicated. The adhesion of human monocytic cell line THP-1 cells to HAECs was determined by adhesion assay. The surface expression of vascular cell adhesion molecule-1 (VCAM-1), endothelial-leukocyte adhesion molecule-1 (E-selectin), and intracellular adhesion molecule-1 (ICAM-1) was measured by cell ELISA. Physiological concentrations of **adiponectin** dose-dependently inhibited TNF-alpha-induced THP-1 adhesion and expression of VCAM-1, E-selectin, and ICAM-1 on HAECs. For the in vivo study, the concentrations of **adiponectin** in human plasma were determined by a sandwich ELISA system that we recently developed. Plasma **adiponectin** concentrations were significantly lower in patients with coronary artery disease than those in age- and body mass index-adjusted control subjects. CONCLUSIONS: These observations suggest that **adiponectin** modulates endothelial inflammatory response and that the measurement of plasma **adiponectin** levels may be helpful in assessment of CAD risk.

L4 ANSWER 10 OF 11 MEDLINE on STN
ACCESSION NUMBER: 1999194557 MEDLINE
DOCUMENT NUMBER: 99194557 PubMed ID: 10092513
TITLE: Paradoxical decrease of an adipose-specific protein, **adiponectin**, in obesity.
AUTHOR: Arita Y; Kihara S; Ouchi N; Takahashi M; Maeda K; Miyagawa J; Hotta K; Shimomura I; Nakamura T; Miyaoka K; Kuriyama H; Nishida M; Yamashita S; Okubo K; Matsubara K; Muraguchi M; Ohmoto Y; Funahashi T; Matsuzawa Y
CORPORATE SOURCE: Graduate School of Medicine, Institute for Molecular and Cellular Biology, Osaka University, 2-2 Yamadaoka, Suita, Osaka, 565-0871, Japan.
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1999 Apr 2) 257 (1) 79-83.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 199905
ENTRY DATE: Entered STN: 19990525
Last Updated on STN: 20000303
Entered Medline: 19990511

AB We **isolated** the human adipose-specific and most abundant gene transcript, apM1 (Maeda, K., et al., Biochem. Biophys. Res. Commun. 221, 286-289, 1996). The apM1 gene product was a kind of soluble matrix protein, which we named **adiponectin**. To quantitate the plasma **adiponectin** concentration, we have produced monoclonal and polyclonal antibodies for human **adiponectin** and developed an enzyme-linked immunosorbent assay (ELISA) system. **Adiponectin** was abundantly present in the plasma of healthy volunteers in the range from 1.9 to 17.0 mg/ml. Plasma concentrations of **adiponectin** in obese subjects were significantly lower than those in non-obese subjects, although **adiponectin** is secreted only from adipose tissue. The ELISA system developed in this study will be useful for elucidating the physiological and pathophysiological role of **adiponectin** in humans.

L4 ANSWER 11 OF 11 MEDLINE on STN
ACCESSION NUMBER: 97103474 MEDLINE
DOCUMENT NUMBER: 97103474 PubMed ID: 8947845
TITLE: **Isolation** and **characterization** of GBP28, a novel gelatin-binding protein **purified** from human plasma.
AUTHOR: Nakano Y; Tobe T; Choi-Miura N H; Mazda T; Tomita M
CORPORATE SOURCE: Department of Physiological Chemistry, School of Pharmaceutical Sciences, Showa University.. yanakano@pharm.showa-u.ac.jp
SOURCE: JOURNAL OF BIOCHEMISTRY, (1996 Oct) 120 (4) 803-12. Journal code: 0376600. ISSN: 0021-924X.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 19970327
Last Updated on STN: 20000303
Entered Medline: 19970317

AB By use of its affinity to gelatin-Cellulofine, a novel protein, GBP28 (gelatin-binding protein of 28 kDa), was obtained from human plasma. GBP28 bound to gelatin-Cellulofine could be eluted with 1 M NaCl. By analysis of its amino-terminal amino acid sequences and the peptides obtained by protease digestion, GBP28 was identified as a novel protein. After repeated gel chromatography of the 1 M NaCl eluate from gelatin-Cellulofine, about 50 micrograms of GBP28 was **purified** from 500 ml of human plasma. On gel chromatography, the protein migrated as a molecule of about 420 kDa. On SDS-PAGE, its molecular mass was 28 kDa under reducing conditions and 68 kDa under nonreducing conditions. Recently, human mRNA specific to adipose tissue, cDNA clone apM1, has been registered [Maeda, K., Okubo, K., Shimomura, I., Funahashi, T., Matsuzawa, Y., and Matsubara, K. (1996) Biochem. Biophys. Res. Commun. 221, 286-289]. The assumed amino acid sequence of cDNA clone apM1 contained all the sequences of GBP28 and its peptides. Therefore, it is evident that the cDNA clone apM1 encodes GBP28 and the protein is specific to adipose tissue. The clone encodes a polypeptide of 244 amino acids with a secretory signal sequence at the amino terminus, a small non-helical region, a stretch of 22 collagen repeats and a globular domain. Thus, GBP28 appears to belong to a family of proteins possessing a collagen-like domain through which they form homo-trimers, which further combine to make oligomeric complexes. Although its biological function is presently unclear, its adipocyte-specific expression suggests that GBP28 may function as an endogenous factor involved in lipid catabolism and storage

or whole body metabolism.

WEST

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Search Results -

Terms	Documents
adiponectin or (adiponectin-like)	19

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L1

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Search History

DATE: Monday, July 21, 2003 Printable Copy Create Case

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DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L1	adiponectin or (adiponectin-like)	19	L1

END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 19 of 19 returned.

☐ 1. Document ID: US 20030108883 A1

L1: Entry 1 of 19 File: PGPB Jun 12, 2003

PGPUB-DOCUMENT-NUMBER: 20030108883
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030108883 A1

TITLE: Methods for identifying compounds that inhibit or reduce PTP1B expression

PUBLICATION-DATE: June 12, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Rondinone, Cristina M.	Libertyville	IL	US	
Trevillyan, James M.	Grayslake	IL	US	
Zinker, Bradley A.	Vernon Hills	IL	US	
Waring, Jeffrey F.	Franklin	WI	US	
Jirousek, Mike	San Diego	CA	US	
Butler, Madeline M.	Santa Fe	CA	US	
Cowsert, Lex M.	Pittsburgh	PA	US	
Monia, Brett P.	Encinitas	CA	US	
Wyatt, Jacqueline	Encinitas	CA	US	

US-CL-CURRENT: 435/6; 435/7.9

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 2. Document ID: US 20030092736 A1

L1: Entry 2 of 19 File: PGPB May 15, 2003

PGPUB-DOCUMENT-NUMBER: 20030092736
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030092736 A1

TITLE: Substituted azole acid derivatives useful as antidiabetic and antiobesity agents and method

PUBLICATION-DATE: May 15, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Cheng, Peter T.	Princeton	NJ	US	
Zhang, Hao	Belle Mead	NJ	US	
Hariharan, Narayanan	Richboro	PA	US	

US-CL-CURRENT: 514/333; 514/340, 514/365, 514/374, 514/396, 546/256, 546/270.4, 546/271.4, 546/272.7, 546/276.4

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☐ 3. Document ID: US 20030044396 A1

L1: Entry 3 of 19 File: PGPB Mar 6, 2003

PGPUB-DOCUMENT-NUMBER: 20030044396
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030044396 A1

TITLE: Methods for treating diseases and increasing longevity

PUBLICATION-DATE: March 6, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Elia, James P.	Scottsdale	AZ	US	

US-CL-CURRENT: 424/93.21; 435/366

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 4. Document ID: US 20020132773 A1

L1: Entry 4 of 19 File: PGPB Sep 19, 2002

PGPUB-DOCUMENT-NUMBER: 20020132773
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020132773 A1

TITLE: Methods for reducing fat by administration of adiponectin

PUBLICATION-DATE: September 19, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Kincade, Paul W.	Nichols Hill	OK	US	
Yokuta, Takafumi	Norman	OK	US	

US-CL-CURRENT: 514/12

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 5. Document ID: US 6582909 B1

L1: Entry 5 of 19 File: USPT Jun 24, 2003

US-PAT-NO: 6582909
DOCUMENT-IDENTIFIER: US 6582909 B1

TITLE: APM1 biallelic markers and uses thereof

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 6. Document ID: US 6566332 B2

L1: Entry 6 of 19 File: USPT May 20, 2003

US-PAT-NO: 6566332
DOCUMENT-IDENTIFIER: US 6566332 B2

TITLE: OBG3 globular head and uses thereof for decreasing body mass

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 7. Document ID: US 6479238 B1

L1: Entry 7 of 19 File: USPT Nov 12, 2002

US-PAT-NO: 6479238
DOCUMENT-IDENTIFIER: US 6479238 B1

TITLE: Polymorphic markers of the LSR gene

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 8. Document ID: US 6344441 B1

L1: Entry 8 of 19 File: USPT Feb 5, 2002

US-PAT-NO: 6344441
DOCUMENT-IDENTIFIER: US 6344441 B1
**** See image for Certificate of Correction ****

TITLE: Lipoprotein-regulating medicaments

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 9. Document ID: JP 2002363094 A

L1: Entry 9 of 19 File: JPAB Dec 18, 2002

PUB-NO: JP02002363094A
DOCUMENT-IDENTIFIER: JP 2002363094 A
TITLE: HEPATIC FIBROGENETIC SUPPRESSOR

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 10. Document ID: JP 2000256208 A

L1: Entry 10 of 19 File: JPAB Sep 19, 2000

PUB-NO: JP02000256208A
DOCUMENT-IDENTIFIER: JP 2000256208 A
TITLE: ANTI-INFLAMMATORY AGENT AND PROPAGATION SUPPRESSOR FOR MONOCYTOID CELL

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

☐ 11. Document ID: WO 3016906 A1

L1: Entry 11 of 19 File: EPAB Feb 27, 2003

PUB-NO: WO0003016906A1
DOCUMENT-IDENTIFIER: WO 3016906 A1
TITLE: METHOD OF DIAGNOSING OR MONITORING SACCHAROMETABOLIC ERROR

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

☐ 12. Document ID: WO 2100427 A1

L1: Entry 12 of 19 File: EPAB Dec 19, 2002

PUB-NO: WO0002100427A1
DOCUMENT-IDENTIFIER: WO 2100427 A1
TITLE: LIVER GENERATION PROMOTER

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

☐ 13. Document ID: WO 2072149 A1

L1: Entry 13 of 19 File: EPAB Sep 19, 2002

PUB-NO: WO0002072149A1
DOCUMENT-IDENTIFIER: WO 2072149 A1
TITLE: METHODS FOR REDUCING FAT BY ADMINISTRATION OF ADIPONECTIN

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

☐ 14. Document ID: WO 2061076 A1

L1: Entry 14 of 19 File: EPAB Aug 8, 2002

PUB-NO: WO0002061076A1
DOCUMENT-IDENTIFIER: WO 2061076 A1
TITLE: ADIPONECTIN-ASSOCIATED PROTEIN

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

☐ 15. Document ID: WO 2003031640 A2

L1: Entry 15 of 19 File: DWPI Apr 17, 2003

DERWENT-ACC-NO: 2003-421278
DERWENT-WEEK: 200339
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TITLE: New primary preadipocyte strain expressing telomerase reverse transcriptase, useful in research applications, screening assays, clinical applications, and in the

administration of therapeutic agents, particularly for obesity

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Drawi Desc | Image

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☐ 16. Document ID: WO 2003016906 A1

L1: Entry 16 of 19 File: DWPI Feb 27, 2003

DERWENT-ACC-NO: 2003-248408
DERWENT-WEEK: 200324
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TITLE: Assay of adiponectin (GBP28) in biological samples for the diagnosis and monitoring of errors of sugar metabolism

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Drawi Desc | Image

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☐ 17. Document ID: WO 2002100427 A1 JP 2002363094 A

L1: Entry 17 of 19 File: DWPI Dec 19, 2002

DERWENT-ACC-NO: 2003-156922
DERWENT-WEEK: 200315
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TITLE: Liver generation promoter comprises adiponectin useful for treating and preventing liver cirrhosis and chronic hepatitis

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Drawi Desc | Clip Img | Image

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☐ 18. Document ID: US 20020132773 A1 WO 200272149 A1

L1: Entry 18 of 19 File: DWPI Sep 19, 2002

DERWENT-ACC-NO: 2003-128066
DERWENT-WEEK: 200312
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TITLE: Method for decreasing fat in adipocytes or the number of adipocytes comprises administration of adiponectin to adipocytes or tissue containing them

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Drawi Desc | Image

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☐ 19. Document ID: WO 200261076 A1

L1: Entry 19 of 19 File: DWPI Aug 8, 2002

DERWENT-ACC-NO: 2002-627480
DERWENT-WEEK: 200267
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TITLE: DNA encoding adiponectin-associated protein which inhibits proliferation of vascular smooth muscle cells, applicable in diagnosis and development of preventives or remedies for arteriosclerosis

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Terms	Documents
adiponectin or (adiponectin-like)	19

Display Format: - Change Format

Previous Page Next Page

1: Circulation. 1999 Dec 21-28;100(25):2473-6. Related Articles, Links

Novel modulator for endothelial adhesion molecules: adipocyte-derived plasma protein adiponectin.

Ouchi N, Kihara S, Arita Y, Maeda K, Kuriyama H, Okamoto Y, Hotta K, Nishida M, Takahashi M, Nakamura T, Yamashita S, Funahashi T, Matsuzawa Y.

Department of Internal Medicine and Molecular Science, Graduate School of Medicine, Osaka University, Osaka, Japan. ouchi@imed2.med.osaka-u.ac.jp

BACKGROUND: Among the many adipocyte-derived endocrine factors, we recently found an adipocyte-specific secretory protein, adiponectin, which was decreased in obesity. Although obesity is associated with increased cardiovascular mortality and morbidity, the molecular basis for the link between obesity and vascular disease has not been fully clarified. The present study investigated whether adiponectin could modulate endothelial function and relate to coronary disease. METHODS AND RESULTS: For the in vitro study, human aortic endothelial cells (HAECs) were preincubated for 18 hours with the indicated amount of adiponectin, then exposed to tumor necrosis factor-alpha (TNF-alpha) (10 U/mL) or vehicle for the times indicated. The adhesion of human monocytic cell line THP-1 cells to HAECs was determined by adhesion assay. The surface expression of vascular cell adhesion molecule-1 (VCAM-1), endothelial-leukocyte adhesion molecule-1 (E-selectin), and intracellular adhesion molecule-1 (ICAM-1) was measured by cell ELISA. Physiological concentrations of adiponectin dose-dependently inhibited TNF-alpha-induced THP-1 adhesion and expression of VCAM-1, E-selectin, and ICAM-1 on HAECs. For the in vivo study, the concentrations of adiponectin in human plasma were determined by a sandwich ELISA system that we recently developed. Plasma adiponectin concentrations were significantly lower in patients with coronary artery disease than those in age- and body mass index-adjusted control subjects. CONCLUSIONS: These observations suggest that adiponectin modulates endothelial inflammatory response and that the measurement of plasma adiponectin levels may be helpful in assessment of CAD risk.

PMID: 10604883 [PubMed - indexed for MEDLINE]

1: Intern Med. 1999 Feb;38(2):202-6. Related Articles, Links

Entrez PubMed

PubMed Services

Related Resources

Role of adipocytokines on the pathogenesis of atherosclerosis in visceral obesity.

Funahashi T, Nakamura T, Shimomura I, Maeda K, Kuriyama H, Takahashi M, Arita Y, Kihara S, Matsuzawa Y.

The Second Department of Internal Medicine, Osaka University Medical School, Suita.

Obesity which is defined as accumulation of excess body fat, is a major cause of atherosclerotic vascular disease in industrial countries. Recent advances in the biology of adipose tissue have revealed that adipose tissue is not simply an energy storage organ but it also secretes a variety of molecules which affect the metabolism of the whole body. Through a systematic search of active genes in adipose tissue, we found that adipose tissue, especially visceral fat expressed numerous genes for secretory proteins (about 30% of total genes analyzed). Among them, plasminogen activator-1 (PAI-1), which is a regulator of the fibrinolytic system, was overexpressed in the visceral fat in an animal model of obesity. Plasma levels of PAI-1 were closely correlated with visceral fat adiposity. Thus, PAI-1 secreted from visceral fat may play some role in thrombotic vascular disease in visceral obesity. Adiponectin, a novel adipose-specific gene product, which has a matrix-like structure, is abundantly present in the bloodstream. Dysregulated secretion of adiponectin may be related to vascular disease in obesity. Biologically active molecules secreted from adipose tissue (adipocytokines) may have important roles in the development of atherosclerotic disease in obesity.

Publication Types:
- Review
- Review, Tutorial

PMID: 10225688 [PubMed - indexed for MEDLINE]

1: Biochem Biophys Res Commun. 1996 Apr 16;221(2):286-9.

cDNA cloning and expression of a novel adipose specific collagen-like factor, apM1 (AdiPose Most abundant Gene transcript 1).

Maeda K, Okubo K, Shimomura I, Funahashi T, Matsuzawa Y, Matsubara K.

Institute for Molecular and Cellular Biology, Osaka University, Suita, Japan.

PMID: 8619847 [PubMed - indexed for MEDLINE]

1: Biochem Biophys Res Commun. 1999 Apr 2;257(1):79-83. Related Articles, Links

Paradoxical decrease of an adipose-specific protein, adiponectin, in obesity.

Arita Y, Kihara S, Ouchi N, Takahashi M, Maeda K, Miyagawa J, Hotta K, Shimomura I, Nakamura T, Miyaoka K, Kuriyama H, Nishida M, Yamashita S, Okubo K, Matsubara K, Muraguchi M, Ohmoto Y, Funahashi T, Matsuzawa Y.

Graduate School of Medicine, Institute for Molecular and Cellular Biology, Osaka University, 2-2 Yamadaoka, Suita, Osaka, 565-0871, Japan.

We isolated the human adipose-specific and most abundant gene transcript, apM1 (Maeda, K., et al., Biochem. Biophys. Res. Commun. 221, 286-289, 1996). The apM1 gene product was a kind of soluble matrix protein, which we named adiponectin. To quantitate the plasma adiponectin concentration, we have produced monoclonal and polyclonal antibodies for human adiponectin and developed an enzyme-linked immunosorbent assay (ELISA) system. Adiponectin was abundantly present in the plasma of healthy volunteers in the range from 1.9 to 17.0 mg/ml. Plasma concentrations of adiponectin in obese subjects were significantly lower than those in non-obese subjects, although adiponectin is secreted only from adipose tissue. The ELISA system developed in this study will be useful for elucidating the physiological and pathophysiological role of adiponectin in humans. Copyright 1999 Academic Press.

PMID: 10092513 [PubMed - indexed for MEDLINE]

1: J Biol Chem. 1995 Nov 10;270(45):26746-9. Related Articles, Links

A novel serum protein similar to C1q, produced exclusively in adipocytes.

Scherer PE, Williams S, Fogliano M, Baldini G, Lodish HF.

PubMed Services

Whitehead Institute for Biomedical Research, Cambridge, Massachusetts 02142-1479, USA.

We describe a novel 30-kDa secretory protein, Acrp30 (adipocyte complement-related protein of 30 kDa), that is made exclusively in adipocytes and whose mRNA is induced over 100-fold during adipocyte differentiation. Acrp30 is structurally similar to complement factor C1q and to a hibernation-specific protein isolated from the plasma of Siberian chipmunks; it forms large homo-oligomers that undergo a series of post-translational modifications. Like adipsin, secretion of Acrp30 is enhanced by insulin, and Acrp30 is an abundant serum protein. Acrp30 may be a factor that participates in the delicately balanced system of energy homeostasis involving food intake and carbohydrate and lipid catabolism. Our experiments also further corroborate the existence of an insulin-regulated secretory pathway in adipocytes.

PMID: 7592907 [PubMed - indexed for MEDLINE]

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